# INHALATION OF HAZARDOUS AIR POLLUTANTS FROM ENVIRONMENTAL TOBACCO SMOKE IN US RESIDENCES

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## ABSTRACT
In the United States, 48 million adults smoke $5 \times 10^{11}$ cigarettes per year. Many cigarettes are smoked in private residences causing regular environmental tobacco smoke (ETS) exposure to at least 31 million nonsmokers (11% of the US population), including 16 million juveniles. ETS contains many chemical species whose industrial emissions are regulated by the US federal government as hazardous air pollutants (HAPs). In this paper, average daily residential exposures to 15 HAPs in ETS are estimated for US nonsmokers who live with smokers. The evaluation is based on material-balance modeling, and utilizes published data on smoking habits, demographics, and housing. Newly measured exposure-relevant emission factors are incorporated. Comparison of exposure concentration estimates with health-based guidelines for chronic exposure suggests that aldehydes — specifically acrolein, acetaldehyde, and formaldehyde — should be of particular concern in ETS. Cumulative population intake results are compared for these compounds against other sources of exposure.

## INDEX TERMS
Environmental tobacco smoke, Exposure, Hazardous air pollutants, Health risk, Mass-balance modeling

## INTRODUCTION
Cigarette smoking is a serious contributor to indoor air pollution. Exposure to environmental tobacco smoke has been linked to an increased risk of many adverse health outcomes, including lung cancer, asthma onset and exacerbation, and acute respiratory illness (NCI, 1999). Concerns about ETS exposure have led to restrictions on smoking in public places, including an almost complete ban on smoking in enclosed workplaces in California. However, regulatory approaches have limited utility for reducing ETS exposures in private residences. Instead, public education, possibly augmented by technological interventions, is best suited to reduce exposures.

Tobacco smoke comprises a large number of chemical constituents, variously partitioned between the gas and condensed phases. Among the constituents of ETS are chemical compounds that are regulated as hazardous air pollutants (HAPs) by the US federal government (USEPA, 2001a). HAPs are species that are known or suspected carcinogens, or that have been shown to cause other serious health effects, such as reproductive problems or birth defects. Characterization and control of HAPs has focused on outdoor sources (USEPA, 2001b). However, because of the close proximity between smokers and nonsmokers, and because of the persistence of pollutants in indoor spaces, the inhalation intake per unit

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emission is 100 or more times higher for ETS than for typical outdoor sources (Smith, 1993; Lai et al., 2000).

To aid in risk assessment, California's Environmental Protection Agency has developed chronic inhalation reference exposure levels (RELs) for 75 individual air toxicants (OEHHA, 2001). A chronic REL represents an estimate of the airborne concentration to which individuals may be indefinitely or routinely exposed with no associated significant health risk. Since ETS is just one of many sources of pollution exposure, chronic exposures associated with ETS near or in excess of the RELs would constitute a cause for concern.

This paper describes an analysis of exposures to specific ETS-constituent HAPs for nonsmokers who live with smokers. Estimated exposure-relevant concentrations are compared to chronic RELs to investigate risks posed by individual HAPs in ETS. The significance of cumulative population exposures is also explored.

## OVERVIEW OF APPROACH

The overall approach adopted here combines a material balance model with published and newly generated data for key input variables. The material balance model is used to estimate exposure concentrations based on data or estimates for these parameters: cigarette consumption patterns, emission factors for HAPs from ETS, residence volume and air-exchange rates, and population statistics. The exposed populations of interest here are nonsmoking adults and all juveniles who live with smokers. A primary goal is to estimate the central tendency of daily exposure to specific hazardous air pollutants by members of this exposed population. The cumulative intake by the entire population will also be estimated. Health risk information will be considered to identify the specific contaminants, among those assessed, which pose the greatest health risk. The health concerns considered here are long-term risks associated with chronic exposure, such as cancer, rather than acute concerns such as odor and irritation.

## CIGARETTE SMOKING HABITS IN THE UNITED STATES

The prevalence of smoking among noninstitutionalized US adults was determined from the Behavioral Risk Factor Surveillance System (BRFSS). This is a state-based, random-digit-dialed telephone survey. Current smokers are defined as those who reported having smoked more than 100 cigarettes during their lifetime and who currently smoke every day or on some days. We combined state-by-state, gender-specific information from BRFSS with census data to estimate that 47.7 million adults (24.9 million males) currently smoke cigarettes in the US (MMWR, 2001; US Census Bureau, 2002), an overall adult smoking prevalence of 22.8%.

The quantity of cigarettes consumed by smokers was estimated from 1997 records reporting an adult, per capita, tax-paid sales rate of 117 packs per year (Tobacco Institute, 1997). Assuming this rate applies to the current US adult population of 213 million, we estimate an annual consumption of about half a trillion ($4.9 \times 10^{11}$) cigarettes by US adult smokers, which corresponds to 1.4 packs per day per smoker.

## HOW MANY PEOPLE ARE EXPOSED TO ETS AT HOME?

We next seek to estimate the number of nonsmokers who are regularly exposed to environmental tobacco smoke (ETS) in their residences. Comprehensive, unbiased estimators do not exist; however, an estimate can be constructed from good proxies. The Third National Health and Nutrition Examination Survey (NHANES III) collected data during 1988-1991 on a nationally representative cross-section of 7079 juveniles (aged 2 mo to 16 y) and 9769 adults (aged 17 and older). In this study, exposure to ETS at home was assumed to occur if

any household member smoked, a condition that was reported for 40.8% of nonsmoking juveniles and 17.4% of nonsmoking adults (Pirkle et al., 1996). This survey likely overestimates the prevalence of household ETS exposure, since it does not exclude homes with smokers who do not smoke indoors. As part of the BRFSS in 1996, data were collected on the prevalence of households with current adult cigarette smokers and any children and adolescents in the home (MMWR, 1997). The same investigation collected information on whether smoking was permitted in some or all areas of the home. From this information, the number of juveniles exposed to ETS at home was estimated to be 15.7 million, or about 22% of all US juveniles. Assuming that the same proportion (22%/40.8% = 0.54) can be applied to the NHANES III adult data, then the proportion of nonsmoking adults regularly exposed to ETS in their own homes is estimated to be 9.4%, which corresponds to 15.6 million people. Thus, we estimate that about 31 million nonsmokers are regularly exposed to ETS in their own homes because they live with smokers, and half of those exposed are children and adolescents.

## EXPOSURE-RELEVANT EMISSION FACTORS

Important input into this analysis is the effective rate at which each of the HAPs is emitted in ETS when a cigarette is smoked. Emission factors have been measured for many ETS constituents in special test chambers. However, recent work indicates that airborne concentrations of some ETS constituents can be greatly affected by sorptive interactions with indoor surfaces (Singer et al., 2002a). Sorption can reduce concentrations and short-term exposures relative to those predicted using an emission factor measured under conditions of low sorption (e.g., an unfurnished metal chamber). We address this issue with the concept of an exposure-relevant emission factor (EREF) that implicitly incorporates sorption effects under realistic furnishing and ventilation conditions. For this analysis, we are interested in ETS concentrations that result from conditions of regular daily smoking. Making use of data from a recent experimental investigation of ETS gas-phase dynamics (Singer et al., 2002b), we derived EREFs by mass balance from gas-phase ETS concentrations measured during the fourth week of an experiment in which 10 cigarettes were smoked each day, 6 days per week, in a furnished 50-$m^3$ room ventilated at 0.6 $h^{-1}$. Calculated EREFs are reported in Table 1.

## QUANTIFYING EXPOSURES TO INDIVIDUAL ETS CONSTITUENTS

In keeping with the goal of providing an assessment that is objective, transparent, and as accurate as the empirical data will support, the daily residential exposure to ETS by nonsmokers is estimated by means of a simple material-balance model. We define the following variables: $N$ is the mean number of cigarettes smoked per day in the home (cig $d^{-1}$); $V$ is the building volume ($m^3$); $A$ is the number of indoor-outdoor air exchanges per day ($d^{-1}$); and $E_i$ is an exposure-relevant emission factor for the HAP of concern (μg $cig^{-1}$). The daily average concentration of the HAP caused by ETS for homes where smoking occurs is estimated as

$$C_i = \frac{NE_i}{AV} \qquad (1)$$

To generate central-tendency estimates for $C_i$, parameter values that approximate the expected mean are selected for terms on the right-hand side of equation (1). We take $N$ = 20 cig $d^{-1}$ (= 1 pack per day) on the basis that people in the US spend an average of 69% of their time indoors at home (Klepeis et al., 2001), half of which is assumed to be awake; the average number of smokers in a home that includes a nonsmoker is 1.4 (Pirkle et al., 1996); and the average cigarette consumption rate is 1.4 packs per smoker per day. The daily number of indoor-outdoor air exchanges is taken to be $A$ = 15 $d^{-1}$, based on a nationwide compilation of

air-exchange rate data (Murray and Burmaster, 1995). The residence volume is taken to be 400 $m^3$, which corresponds approximately to the median floor area (1730 $ft^2$) reported for US single-family detached and mobile homes (US Census Bureau, 2001).

To facilitate our analysis, we assume that the average concentration of an ETS constituent in air breathed by the nonsmoker over the course of a day, caused by smoking in their home, is the same as the average in ventilation air that passes through the residence. This is approximately the condition that would obtain if the nonsmoker were present in the home during, and for several hours after cigarette consumption by the smoking resident(s). To the extent that nonsmokers are not present during and after smoking, the exposure would be reduced. Conversely, if the nonsmoker were present in the same room as the smoker during smoking events, the exposure concentration would probably be higher than estimated here. Table 1 presents an estimate of daily average exposure concentrations for 15 HAPs and compares these results with chronic RELs, where those are available. The rightmost column of Table 1 shows the ratio of estimated exposure concentration to the chronic REL. The highest value of this ratio, ~ 30, is found for acrolein. Values in the vicinity of 1 are found for the other aldehydes: acetaldehyde and formaldehyde. That these ratios are of order one or greater than one, and that these are the highest ratios, suggests that, among the compounds considered here, exposure to aldehydes from ETS should be of particular concern.

**Table 1.** Central-tendency estimates of daily-average exposure concentration to HAPs in ETS for nonsmokers who live with a smoker.

| Compound | EREF ($\mu g\ cig^{-1}$) [a] | Exposure conc. ($\mu g\ m^{-3}$) | Chronic REL ($\mu g\ m^{-3}$) [b] | Exposure/REL |
|---|---|---|---|---|
| **Acetaldehyde** | **2267** | **7.6** | **9** | **0.8** |
| Acetonitrile | 1028 | 3.4 | na | — |
| **Acrolein** | **560** | **1.9** | **0.06** | **31** |
| Acrylonitrile | 181 | 0.6 | 5 | 0.1 |
| Benzene | 417 | 1.4 | 60 | 0.02 |
| 1,3-Butadiene | 461 | 1.5 | 20 | 0.08 |
| 2-Butanone | 300 | 1.0 | na | — |
| Cresol isomers | 70 | 0.2 | 600 | 0.0003 |
| Ethylbenzene | 136 | 0.5 | 2000 | 0.0002 |
| **Formaldehyde** | **979** | **3.3** | **3** | **1.1** |
| Methylnaphthalenes [c] | 52 | 0.2 | na | — |
| Naphthalene | 42 | 0.1 | 9 | 0.02 |
| Phenol | 162 | 0.5 | 200 | 0.003 |
| Styrene | 169 | 0.6 | 900 | 0.0006 |
| Toluene | 879 | 2.9 | 300 | 0.01 |

[a] Exposure-relevant emission factor; see text for details.
[b] Reference exposure level for chronic conditions (OEHHA, 2001).
[c] Sum of 1-methylnaphthalene and 2-methylnaphthalene isomers.

## INHALATION INTAKE OF ALDEHYDES FROM ETS

In this section, the total annual inhalation intake of ETS-associated aldehydes by US nonsmokers who live with smokers is estimated. To put the result in context, an estimate is also made of the total inhalation intake by all US residents from all primary outdoor emissions.

For the 31 million nonsmokers who live with smokers, the estimated ETS-associated exposure concentration of acrolein is 1.9 μg $m^{-3}$. The average volume of air breathed per day is 12 $m^3$ (Layton, 1993). Multiplying these three numbers together and then converting to mass inhaled per year, we estimate the total intake of acrolein by this population to be 260 kg $y^{-1}$. This corresponds to about 0.1% of the total of 280 tonnes of acrolein generated in ETS each year.

The total primary emissions of acrolein to the atmosphere from all sources in the United States are about 26,000 tonnes per year, according to the Toxic Release Inventory (USEPA, 2001b). Most of this is from area sources that would tend to occur in populated regions. The fraction of pollution emitted to urban air that is breathed by people has been estimated to lie in the range 0.7-70 per million, depending on meteorology and on the size of the populated region, and assuming a typical urban population density of 1000 $km^{-2}$ (Lai et al., 2000). Taking 10 per million as a crude estimate of the average condition in the United States, we estimate that the total inhalation intake of acrolein from all ambient emission sources is ~ 300 kg $y^{-1}$. Thus, although ETS is almost negligible as a source of urban air concentrations of acrolein, it contributes as much to cumulative human intake in residential settings alone as do all sources of emissions to the ambient air. For acetaldehyde, a similar conclusion is reached: ETS in homes contributes about as much to human inhalation intake (~ 1000 kg/y) as do all primary-emission sources to ambient air. On the other hand, formaldehyde emissions to ambient air are estimated to be stronger contributors to human inhalation exposure (~ 3000 kg/y) than ETS in homes (~ 400 kg/y).

## CONCLUSIONS

Most health-risk evaluations of exposure to environmental tobacco smoke are based on epidemiological investigations that use questionnaires or marker compounds (e.g., cotinine in body fluids) to estimate exposure. The approach presented here is complementary: it can help to identify specific compounds in ETS that contribute significantly to the overall health risks. The results of our study indicate that the aldehydes in ETS, especially acrolein, should be of particular concern as contributors to health risk from chronic, residential ETS exposure. Future studies may usefully be focused on better characterizing exposure to aldehydes from ETS and on the effectiveness of intervention measures to reduce such exposures.

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